Insomnia is a feature of many psychiatric disorders such as depression, post-traumatic stress disorder (PTSD), borderline personality disorder and anxiety disorders. In recent years, brain imaging studies have revealed brain structural changes in people with psychiatric disorders. As a result, scientists have long believed that both the insomnia and the brain structural changes were related to the psychiatric disorder. Recent research now indicates that the brain structural changes may be related only to the insomnia.

Difficulty initiating or maintaining sleep – in other words, insomnia – is usually the result of (i.e., secondary to) another problem such as a psychiatric disorder, an adverse drug effect, a sleep disorder (e.g., sleep-disordered breathing, circadian rhythm sleep disorders, restless legs syndrome, periodic limb movements, narcolepsy and parasomnias), pain, or poor sleep hygiene. Insomnia is considered transient if it lasts less than one month and chronic if it lasts more than one month. Insomnia that is not secondary to another condition is called primary insomnia. In people with primary insomnia, the quality of the sleep is nonrestorative and interferes with occupational, educational or social functioning.

Insomnia in some cases can result in sleep deprivation since the amount of sleep during a sleep period is shortened by delays in getting to sleep or early arousals from sleep. Animal studies indicate that long-term sleep deprivation decreases the development (and therefore the volume) of the hippocampus. Behavioral studies on people who have been sleep deprived for short periods of time indirectly suggest that a similar structural change may be occurring in the human brain. For example, people who are sleep deprived have impairments in memory, indicating impaired function of the hippocampus (which in turn may be a reflection of hippocampal structural changes). However, it is not possible to experimentally sustain sleep deprivation in humans for periods sufficient enough to demonstrate whether sleep deprivation induces brain structural changes. Viewing primary insomnia as a useful human model to test the hypothesis that chronically disturbed sleep is associated with changes in brain structure, Dieter Riemann and colleagues used magnetic resonance imaging (MRI) to measure the total brain volume and the volume of certain regions of the brains of people who have primary insomnia. Riemann suspected that brain structural changes in people with primary insomnia would be similar to changes that had been noted in animals subjected to chronic sleep deprivation.

The study involved eight subjects with primary insomnia and eight controls (i.e., good sleepers). The insomnia subjects reported a worse sleep quality than did the controls, as assessed by the Pittsburgh Sleep Quality Index (PSQI). The average PSQI score of the insomnia subjects was 11.1, while the average score of the controls was 3.5. (A score over 5 indicates poor sleep quality.) None of the study participants had any current or lifetime psychiatric disorders.

MRI scans detected no brain pathology among the study participants. From the scans, Riemann and colleagues measured each study participant’s total brain volume and the volume of the following brain areas: the prefrontal cortex, the orbitofrontal cortex, the anterior cingulate cortex, the amygdala, and the hippocampus. These structures play a role in memory, affect and decision-making.

Riemann found that subjects with primary insomnia had a significantly smaller hippocampal volume than the controls. Since the subjects had no psychiatric disorders, Riemann concluded that the brain structural changes are associated with insomnia alone and that insomnia may either result from, or contribute to, brain structural changes.

Ellenmarije Altena and colleagues in their recent study were the first scientists to demonstrate that the magnitude of brain structural changes is associated with the severity of insomnia. The study involved 24 subjects (who had struggled with primary insomnia on average for 17 years) and 13 controls. Compared with the controls, the subjects had significantly more complaints of insomnia (or other sleep problems), somatic complaints, and complaints of reduced vitality and social functioning, as assessed by the PSQI, the Symptoms Checklist 90 (SCL 90), and the Sleep Disorders Questionnaire (SDQ)–insomnia scale.

All study participants underwent MRI scanning. The scans revealed that, compared with the controls, the subjects with primary insomnia had a smaller volume of gray matter in certain areas of the orbitofrontal cortex (which plays a role in emotion, decision-making, and problem-solving), the parietal cortex (which plays a role in somatosensory function), and the occipitoparietal cortex (which plays a role in mental imagery and episodic memory retrieval - also called "autobiographical memory").

Altena theorizes that decreased gray matter in the parietal cortex may impact a person’s ability to disengage from external stimuli. This finding could explain the great difficulty people with insomnia have in entering a state of rest sufficiently to allow sleep. Altena also believes that decreased gray matter in the orbitofrontal region may explain the difficulty people with insomnia have with decision-making, problem-solving and emotional control.

Within the insomnia group, Altena noted there was a strong negative correlation between the severity of insomnia symptoms and the gray matter volume of the left orbitofrontal cortex; in other words, the gray matter volume decreased as the severity of insomnia increased (as measured by the SDQ–insomnia score). There was no correlation between the severity of insomnia and the gray matter volume of the parietal areas (i.e., occipitoparietal and parietal cortices).

Altena did not find a reduced hippocampal volume as had
Riemann. However, discrepancies between the results of the two studies may have been related to normal age-related changes in the medial temporal region (i.e., the inner portion of the temporal lobe) where the hippocampus is located. The age of the subjects in the Riemann study was on average 48 years old, while participants in the Altena study were on average 60 years old.

Current treatments for insomnia often focus on alleviating contributing factors to the difficulty sleeping. For example, behavioral changes such as avoiding caffeinated drinks, minimizing stimulating activities before bedtime, or establishing a calming ritual before bedtime may be tried to improve sleep hygiene. Cognitive-behavioral therapy also may be used as a primary treatment of insomnia. If these treatments are ineffective, then a sleep study may be performed to evaluate for an underlying sleep disorder such as obstructive sleep apnea, periodic limb movements or parasomnias.

If behavioral approaches fail or a cause cannot be determined for the insomnia, then medications may be prescribed to induce sleep. Many hypnotic (i.e., sleep-inducing) drugs – such as the benzodiazepines (e.g., flurazepam) and certain nonbenzodiazepine drugs (e.g., zolpidem, eszopiclone, and zaleplon) – enhance the actions of the inhibitory neurotransmitter gamma-amino butyric acid (GABA) in the brain. The neuronal inhibition slows the neuronal firing rate, thereby inducing sleep. Cognitive-behavioral therapy also should be mentioned as a primary treatment of insomnia.

The long-term use of some hypnotic drugs can lead to physiological dependence. As a result, increasingly larger doses are needed to accomplish the same hypnotic effect. For this reason, hypnotics typically are not prescribed for a long period of time. Rebound insomnia can occur with the sudden discontinuation of some hypnotic drugs in people who have taken a hypnotic drug for a long period of time. The consequences of dependence and rebound insomnia on occupational, educational, and social functioning may be problematic for some people with primary insomnia for whom drug therapy may be the only effective sleep-inducing treatment.

Inhibited neuronal growth or loss of neurons can result in the decreased volume of a brain structure. This reduction may then impact the function of that structure. For example, the loss of neurons may impact the transmission of neurotransmitters in affected structures. In support of this, some research has found that the brain level of GABA in people with primary insomnia is lower than in people without the disorder. Although it is possible that the reduced GABA level results from the reduced production of GABA, it is also possible that the reduced GABA level results from a less-than-normal amount of or loss of GABA-containing neurons.

Future studies are needed to determine whether the volume reduction of the hippocampus, gray matter, or other brain structures is clearly related to insomnia and if so, whether the brain changes are preceded by or are induced by insomnia. If brain structural changes are induced by insomnia, then diagnosing and treating insomnia earlier may avoid creating adverse brain structural changes that could ultimately maintain or exacerbate the insomnia. If brain structural changes precede insomnia, then detecting and understanding the causes of these changes earlier could limit subsequent neuronal damage and potentially delay or prevent future insomnia problems.

REFERENCES


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